WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 6, 1591-1616. Review Article ISSN 2277–7105

A COMPREHENSIVE CONCEPTUAL AND PHARMACOLOGICAL EVALUATION OF KITIBHA KUSHTA AND ITS THERAPEUTIC MODALITIES IN AYURVEDA WITH SPECIAL REFERENCE TO PLAQUE PSORIASIS - AN INTEGRATIVE REVIEW

[1]*Dr. Anusha M. Janna, [2]Dr. Poornima B.

[1]Post Graduate Scholar, Department of Post Graduate Studies in Kayachikitsa.

[2]Professor, Department of Post Graduate Studies in Kayachikitsa. Hubli Ayurveda Seva Samiti's, Ayurveda Mahavidyalaya and Hospital, Hubballi, Karnataka.

Article Received on 15 Feb. 2026,
Article Revised on 05 March 2026,
Article Published on 16 March 2026,

https://doi.org/10.5281/zenodo.19086806

***Corresponding Author**

Dr. Anusha. M. Janna

Post Graduate Scholar, Department of Post Graduate Studies in Kayachikitsa.

How to cite this Article: [1]*Dr. Anusha. M. Janna, [2]Dr. Poornima B. (2026). A Comprehensive Conceptual And Pharmacological Evaluation of Kitibha Kushta and Its Therapeutic Modalities In Ayurveda With Special Reference To Plaque Psoriasis - An Integrative Review. World Journal of Pharmaceutical Research, 15(6), 1591–1616.

ABSTRACT

Background: Plaque psoriasis is a chronic immune-mediated inflammatory dermatosis affecting approximately 2–3% of the global population and significantly impairing quality of life. In Ayurveda, similar clinical features are described under *Kitibha Kushta*, a *Vata–Kapha* predominant *Tridoṣhaja* disorder involving *Tvak, Rakta, Mamsa,* and *Lasika*. **Objective:** To critically evaluate the Ayurvedic concept of *Kitibha Kushta* and correlate its etiopathogenesis, clinical features, and therapeutic principles with contemporary understanding of plaque psoriasis. **Methods:** A narrative review of classical Ayurvedic texts (*Brihattrayi* and *Laghutrayi*) was undertaken to analyse *Nidana, Samprapti, Lakshana,* and *Chikitsa*. Relevant biomedical literature was reviewed to examine the immunological and molecular mechanisms of psoriasis and the pharmacological actions of selected Ayurvedic drugs. **Results:** The Ayurvedic pathogenesis involving *Agnimandya, Ama formation, Rasa Rakta srotodushti,* and *Dosha–Dushya sammurchana* parallels modern concepts of metabolic inflammation, oxidative stress, keratinocyte hyperproliferation, and Th17-axis activation (TNF-α, IL-17, IL-23). Classical features such as *Shyava varna, Khara/Parusha sparsha,* and *Ugra kandu* closely resemble psoriatic plaques. Therapeutic measures including *Nidana parivarjana,*

Shodhana (Virechana, Raktamokshana), Shamana, and *Rasayana* demonstrate multi-target actions. Phytoconstituents such as curcumin, psoralen, nimbolide, guggulsterone, catechins, fulvic acid, and sulphur derivatives exhibit NF-κB, STAT3, and MAPK inhibition with cytokine suppression and antioxidant effects. PASI scoring provides an objective tool for integrative assessment. **Conclusion:** Significant conceptual and mechanistic alignment exists between *Kitibha Kushta* and plaque psoriasis. Ayurvedic management offers a holistic, multi-target approach that warrants further systematic clinical validation.

KEYWORDS: *Kushta, Kitibha Kushta,* Plaque Psoriasis, PSAI Scale.

1. INTRODUCTION

Skin diseases are among the leading causes of non-fatal morbidity worldwide, with psoriasis affecting nearly 2–3% of the global population and significantly impairing quality of life. It is a chronic, immune-mediated inflammatory disorder characterized by erythematous, scaly plaques and a relapsing course, often associated with metabolic and psychosocial comorbidities. Although topical agents, phototherapy, systemic immunosuppressants, and biologics provide symptomatic control, their long-term use is limited by adverse effects, high cost, and frequent recurrence, necessitating safer and more sustainable therapeutic approaches.

In Ayurveda, dermatological disorders are described under *Kushta*, a group of chronic and recurrent diseases involving vitiation of *Tridosha* and structural impairment of *Tvak, Rakta, Mamsa,* and *Lasika*. *Kitibha Kushta*, classified under *Kshudra Kushta*, presents with *Shyava varna*, *Khara/Parusha sparsa*, and *Ugra kandu*, indicating *Vata–Kapha* predominance and showing close clinical resemblance to plaque psoriasis. Its pathogenesis involves *Agnimandya*, *Amotpatti*, *Rakta dushti*, and *Dosha–Dushya sammurchana* localized in the skin.

Ayurvedic management emphasizes *Shodhana, Shamana,* external therapies, and *Nidana parivarjana*, aiming at correction of the underlying pathology and prevention of relapse. However, systematic correlations between classical descriptions of *Kitibha Kushta* and contemporary clinical parameters of psoriasis remain limited. The present study critically evaluates *Kitibha Kushta* in the context of plaque psoriasis to explore the relevance of Ayurvedic principles in its comprehensive management.

1.1. Study objectives

To evaluate *Kitibha Kushta* through classical Ayurvedic parameters and correlate its clinical features, pathogenesis, and therapeutic principles with plaque psoriasis to assess the scope of Ayurvedic management in its long-term care.

SPECIFIC OBJECTIVES

1. To analyse the classical descriptions of *Kitibha Kushta* with respect to *Nidana, Samprapti, Laksana, and Chikitsa* from major Ayurvedic texts.
2. To correlate the clinical features of *Kitibha Kushta* with the dermatological presentation of plaque psoriasis.
3. To assess the *Dosha–Dushya involvement* in *Kitibha Kushta* and map it with the contemporary understanding of psoriasis pathophysiology. The role of *Rakta dushti, Agnimandya, and Ama* in the manifestation and chronicity of the disease.
4. To examine the therapeutic utility of *Shodhana* and *Shamana chikitsa*, along with *Bahirparimarjana* measures, in the management of *Kitibha Kushta*.
5. To review the importance of *Nidana parivarjana and Pathya–apathya* in preventing recurrence.
6. To explore the feasibility of using PASI score as an objective assessment tool for Ayurvedic management of *Kitibha Kushta*.

2. AYURVEDIC CONCEPTUAL FRAME WORK

2.1. *Kitibha Kushta*: classical definition

In Ayurvedic literature, *Kushta* is used as a collective term encompassing a wide spectrum of skin disorders. The term *Kushta* is derived from the root word "*kush*", which denotes the manifestation of pathology originating internally and presenting externally on the skin. *Kushta* is considered one of the *Ashta Mahagada*, signifying a group of diseases that are difficult to cure. The classical statement "*kaalenopekṣitam yasmat sarvaṃ kuṣṇati tad vapuh*" implies that, if neglected over time or left untreated, *Kushta* progressively affects the entire body, leading to disfigurement and an unsightly appearance.[1] The pathogenesis of *Kushta* involves seven factors (*sapta dravya*): *Tridosha, Tvak, Rakta, Mamsa,* and *Lasika*. *Kushta* is classified based on severity, *Dosha* predominance, and clinical presentation. *Kitibha Kushta* is included under *Kshudra Kushta* by Acharya Charaka[2], Sushruta[3], Vagbhaṭa[4], and Madhava Nidana.[5]

Kustha is broadly classified into *Mahakushta* and *Kshudra Kushta*. *Kitibha Kushta* is categorized under *Kshudra Kushta*. The term *Kitibha*, as explained in Sabdakalpadruma, is

derived from "*kitiriva bhati*", signifying lesions resembling kiti. The word "*kiti*" denotes small organisms such as lice or bugs, while "*bha*" refers to color, collectively indicating the characteristic appearance of the disease. *Kitibha Kushta* presents with characteristic symptoms such as ***Shyava Varna*** (blackish discoloration), ***Kinakhara Sparsha*** (rough texture), ***Parusha*** (dryness), and ***Ugra Kandu*** (intense itching). It is classified as a *Tridoshaja Vyadhi* with predominant involvement of *Vata* and *Kapha dosha*.

2.2. *Nidana* (causative factors)

Ayurvedic classics describe general causative factors (*Samanya Nidana*) and for all varieties of *Kushta* rather than specifying separate etiological factors for each type. Hence, the etiological factors of *Kushta*, including *Kitibha Kushta*, can be broadly classified into the following categories:

- ***Ahara Hetu*** **(dietary factors)**

Viruddha Ahara	**C.S**	**Su.S**	**A.S**	**A.H**	**B.S**	**H.S**	**M.N**	**B.P**
Intake of Mulaka, Lashuna etc with milk	+	+	+	+	+	+	+	+
Gramya, Anupa, Audaka Mamsa with milk	+	-	-	+	-	-	-	-
Use of Matsya, Nimba and Ksheera together	-	-	-	-	+	-	-	-

Mithya Ahara	**C.S**	**Su.S**	**A.S**	**A.H**	**B.S**	**H.S**	**M.N**	**B.P**
Adhyasana	+	+	-	-	-	+	+	+
Vishamasana	+	+	-	-	-	-	-	-
Asatmya Ahara	-	+	-	-	-	-	-	-
Intake of Ahara during Ajeerna	+	+	-	-	-	-	+	+
Excessive Snehana	+	-	-	-	-	-	-	-

- ***Vihara Hetu*** **(lifestyle and behavioral factors)**

Mithya vihara	**C.S**	**Su.S**	**A.S**	**A.H**	**B.S**	**H.S**	**M.N**	**B.P**
Shitoshna Vyatyasa sevana and anupurvya sevana	+	-	-	-	+	-	-	+
Use of Santharpana and Apatarpana diet without Sequence	+	-	-	-	-	-	-	+
Practice of physical exercise and sunbath after heavy meals	+	-	-	-	-	-	+	+
Suppression of Chardi, Mutra, Purisha like Vegas	+	+	-	-	+	-	+	+
Divaswapna after lunch	+	-	-	-	+	-	-	-

- ***Achara*** **and** ***Manasika Hetu*** **(psychological and ethical factors)**

- *Papa Karma*
- *Vipra Guru Tiraskara*
- *Sadhu Ninda*
- *Chinta*
- *Bhaya, Krodha, Shoka*

Other *Nidanas* (miscellaneous causes) *Samsarga Hetu*[6] (Miscellaneous Causes)

Susruta and Vagbhata refer *Kushta* as an *Aupasargika Roga* (communicable disease). The disease can be transmitted through close physical touch (*Prasanga*), (*Gatrasamsparsa*), (*Nisvasat, Sahabhojanat*), sharing food, bed or clothing, and extended contact. These observations suggest that *Kushta* has characteristics similar to infectious or transmissible diseases.

***Kulaja Nidana*[7] (Hereditary Etiology)**

Kulaja Nidāna, also referred to as *Anuvamsika Nidana*, denotes the hereditary basis of *Kushta*. Susruta has classified *Kushta* under *Adibala-pravrtta Vyadhi*, indicating diseases arising from defects in the parental reproductive elements *(Sukra* and *Shonita*). Such defects lead to a predisposition for *Kushta*, which may be transmitted from affected parents to their offspring. This concept reflects an early understanding of genetic or congenital susceptibility in the pathogenesis of *Kushta*.

***Krimi Hetu*[8,9] (Microbial / Infective Etiology)**

Acharya Susruta stated that all varieties of *Kushta* arise due to the vitiation of *Vata, Pitta, Kapha*, and *Krimi*. Similarly, Caraka has indicated that the etiological factors and therapeutic principles described for *Raktaja Krimi* are applicable to *Kushta*, suggesting that *Krimi* (microbial or parasitic factors) play a significant role in its pathogenesis. Hence, *Krimi* can be considered one of the important causative factors in *Kitibha Kushta*.

***Chikitsa Vibhramsa Hetu* (Therapeutic Errors and Disease Progression)**

In the early stages of diseases such as *Raktaarsha*[10], *Raktapitta*[11], and *Amatisara*[12], inappropriate therapeutic approaches—particularly the use of *Stambhana* (suppressive therapy)—may lead to *Dosha Utklesha* and *Tiryag Gati* (abnormal movement and aggravation of doṣas). This pathological diversion facilitates deeper tissue involvement and thereby contributes to the development of *Kushta*.

Role of *Rakta* in the Pathogenesis of *Kushta*

Classical texts describe *Kushta* as a *Rakta-pradoshaja* and *Santarpanajanya* disorder, indicating that vitiation of blood (*Rakta*) along with over-nutrition and metabolic derangement plays a pivotal role in disease manifestation. Therefore, *Rakta dushti* and *Santarpana nidana* are considered key pathogenic factors in the development of *Kushta*.

2.3. *Purvarupa and Rupa* (Prodromal and Clinical Features)

The prodromal (*Purvarupa*) and clinical manifestations (*Rupa*) of *Kitibha Kushta* are not described separately in the classical Ayurvedic texts. Hence, the general prodromal and clinical features of *Kushta* are considered applicable to *Kitibha Kushta*. In milder forms, the characteristic features of *Kitibha* are included under the broader symptomatology of *Kushta*.

2.4. Lakshana of *Kitibha Kushta* According to different Acharyas

Lakshana	C.S	Su.S	A.H	B.S	M.N	YR	B.P	Ka.S
Rooksham	-	-	+	-	-	-	-	-
Kinakhara Sparsha	+	-	+	-	+	+	+	-
Kandu	-	+	+	-	-	-	-	-
Parusham	+	-	+	-	+	+	+	-
Asitam	-	-	+	-	-	-	-	-
Khara sparsham	+	-	+	-	+	+	+	-
Shyavam	+	-	+	-	+	+	+	-
Vrittam	-	+	-	+	-	-	-	-
Ghanam	-	+	-	+	-	-	-	-
Sraavi	-	+	-	+	-	-	-	-
Snigdam	-	+	-	+	-	-	-	-
Krishnam	-	+	-	+	-	-	-	-
Punah Prasravati	-	-	-	+	-	-	-	-
Guru	-	-	-	-	-	-	-	+
Vardatecha Samutpannam	-	-	-	+	-	-	-	+
Arunam	-	-	-	-	-	-	-	+
Prashanthnicha punarutpadyate	-	-	-	-	-	-	-	+
Vridhimanti	-	-	-	-	-	-	-	+

Ayurvedic Contemporary understanding

Ayurvedic Term	Literal Meaning	Modern Dermatological Correlate
Rookṣham	Dryness	Xerosis due to impaired epidermal barrier and reduced lipid content
Kinakhara Sparsha	Rough to touch	Hyperkeratosis with scaling plaques
Kaṇḍu	Itching	Pruritus mediated by inflammatory cytokines and neural sensitization
Parusham	Hard/harsh texture	Epidermal thickening (acanthosis)
Asitam / Krshnam	Dark/blackish discoloration	Post-inflammatory hyperpigmentation or dusky plaques
Khara Sparsham	Coarse surface	Thick adherent scales (parakeratosis)

Shyavam	Dusky/violaceous color	Chronic inflammatory plaque with altered vascularity
Vrittam	Circular lesions	Well-demarcated round plaques
Ghanam	Thickened lesion	Plaque elevation due to keratinocyte hyperproliferation
Sraavi	Oozing (in some stages)	Serous exudation in inflamed or fissured plaques
Snigdam	Greasy appearance (occasionally)	Oily scale in certain anatomical sites (sebopsoriasis)
Punah Prasravati	Recurrent discharge	Recurrent inflammation with fissuring/exudation
Guru	Heaviness/thick feel	Indurated plaques with dermal inflammation
Vardatecha Samutpannam	Progressive enlargement	Peripheral plaque expansion (active margins)
Arunam	Reddish discoloration	Erythema from dilated dermal capillaries
Prashanthnicha Punarutpadyate	Recurs after remission	Relapsing–remitting course
Vrdhimanti	Increase in number/size	Plaque multiplication and coalescence

2.5. *Samprapti* (Pathogenesis)

Samprapti is a key component of *Nidana Pancahaka* that explains the sequential development of a disease from its causative factors to clinical manifestation. It provides a clear understanding of the involvement of *Dosha, Dhatu, and Srotas*, thereby forming the basis for accurate diagnosis and rational therapeutic planning.

According to Acharya Charaka, exposure to etiological factors leads to the simultaneous vitiation of *Tridosha*, which subsequently affects *Tvak, Rakta, Mamsa*, and *Lasika*. This pathological involvement results in *Saithilyata* (loss of integrity) of these *Dhatus*, creating a conducive environment for the manifestation of *Kushta*. Thus, *Kushta* is understood as a disease arising from the combined derangement of *Dosha* and *Dhatu* with structural and functional impairment of the skin and underlying tissues.

Acarya Susruta describes that *Nidana sevana* leads to the vitiation of *Vata*, which in association with *Kapha* disturbs *Pitta* and enters the *Tiryak Siras*. The vitiated *Doshas* are then deposited in the skin, producing *Mandalas* (lesions); if untreated, they progressively infiltrate deeper *Dhatus*.

***Vishesha Samprapti:* Contemporary Ayurvedic understanding[13]**

Ayurvedic Samprapti	Conceptual Meaning	Modern Psoriasis Correlation

Nidana Sevana	Viruddhahara, guru-snigdha ahara, divasvapna, stress → dosa prakopa	Genetic predisposition + environmental triggers (stress, obesity, drugs, infection, alcohol, smoking)
Jatharagni & Dhatvagni Mandya	Impaired digestion & metabolism	Metabolic dysfunction, gut–skin axis disturbance, altered microbiome, systemic inflammation
***Ama* Formation**	Toxic, pro-inflammatory metabolic by-products	Chronic systemic inflammation, oxidative stress, circulating inflammatory mediators
Ama* + *Tridosha Dushti	Vata–Kapha predominance with Pitta involvement	Immune dysregulation with Th1/Th17 activation, cytokine storm (TNF-α, IL-17, IL-23)
Rasarakta-vaha Srotodushti	Microcirculatory and nutritive channel impairment	Dermal vascular proliferation, endothelial activation, increased skin blood flow
***Tvak, Rakta, Mamsa, Lasika* Involvement**	Multi-tissue pathology of skin and underlying dhatus	Keratinocyte hyperproliferation, dermal inflammation, immune cell infiltration, lymphatic involvement
Dosha–Dushya Sammurchana	Pathogenic interaction at tissue level	Cytokine-driven keratinocyte–immune cell feedback loop
Manifestation of Kitibha Lakshanas	*Shyava Varna, Kinakhara Sparsha, Parusha, Ugra Kandu.*	Blackish discoloration, rough texture, dryness, intense itching

2.6. Samprapti Ghatakas

1. ***Dosha****-Vata kapha pradhana, Tridosha*
2. ***Dushya****- Rasa, Rakta, Mamsa, Lasika*
3. ***Agni*** *-Jataragni and Dhatwagni*
4. ***Ama*** *-Jataragni Mandya janya and Dhatwagni Mandya Janya Ama*
5. ***Srotas*** *- Rasavaha, Raktavaha Mamsavaha*
6. ***Srotho dushti Prakara*** *-Sangha, Vimargagamana*
7. ***Udbhava Sthana*** *-Amashaya*
8. ***Adhishtana*** *-Twak, Rakta. Mamsa and Lasika*
9. ***Vyaktha Sthana****- Twak*
10. ***Sanchara Sthana*** *- Sarva Sharira*
11. ***Roga Marga*** *- Bahya roga Marga*
12. ***Roga Swabhava*** *- Chirakari*

2.7. Sadhya-Asadhyata

According to Acharya Charaka, assessment of *Sadhya-Asadhyata* (progonosis) is crucial in the

management of *Kushta*. Patients who are weak and exhibiting all the classical lakshanas of *kushta* are considered *Asadhya* and should not be treated. *Kushta* associated with *Vata-Kapha* predominance and whereas lesions dominant by single dosa are regarded as *Sadhya*. Conversely, *Kushta* involving multiple *doshas Kapha-Pitta, Vata -Pitta* and deeper *Dhatus* are considered *Kruchra Sadhya*.

Based on the above considerations, *Kitibha Kushta* of early origin with predominance of *Vata–Kapha Dosha* and involvement limited to *Rasa, Rakta and Mamsa Dhatus* is considered *Sadhya*. However, chronic *Kitibha Kushta* associated with *Tridosha* vitiation and deeper *Dhatu* involvement is regarded as *Yapya* or *Asadhya.*

2.8. Chikitsa

To study the treatment of *Kushta* systemically, it is necessary to look at three principles of treatment i.e. 1) *Nidana Parivarjana*, 2) *Samshodhana, 3) Samshana.*

Nidana Parivarjana

Nidana Parivarjana refers to avoidance of etiological factors and helps to arrest further progression of *Kushta* by preventing *Dosha* vitiation. *Mithya Ahara-Vihara* and *Viruddha Ahara* are the major causative factors and should therefore be avoided. Acharya Charaka defines *Pathya* as wholesome diet and regimen beneficial to body and mind, while those that are harmful are termed *Apathya*. These principles are applicable to *Kitibha Kushta* as well.

Samshodana

Shodhana is the therapy aimed at eliminating vitiated *Doshas* from the body, thereby enhancing the efficacy of subsequent treatments. All Acharyas have emphasized *Sodhana* in the management of *Kushta,* as the disease is characterized by extensive *Dosha* vitiation (*Bahu-doshatva*).[14] *Kushta* is a *Tridoshaja Vyadhi*[15] in which the *Doshas* exhibit *Tiryag-gati*[16], making it inherently difficult to cure (*Duschikitsya*). However, by removing the root cause through *Sodhana,* the disease becomes more amenable to treatment.

In accordance with the predominance of the involved *dosha* and the rogi bala (patient's strength), appropriate *shodhana* procedures such as *vamana* (Emesis), *virechana* (Purgation), *raktamokshaṇa* (Bloodletting), Basthi (Enema), *nasya* (Intranasal Administration) and Dhumapana (Fumigation) should be selected.

According to Caraka and Vagbhata, *Sodhana* should be selected based on the predominant

Dosha—Ghritapana for *Vata*, *Vamana* for *Kapha*, and *Virecana* along with *Raktamokshana* for *Pitta*. In cases of severe *Dosha* vitiation, repeated *Sodhana* is recommended at regular intervals. Susruta further advocates *Ubhayata Samsodhana* even in the *Purvarupa* stage and recommends *Samsodhana* in *Rasa-gata*, *Rakta-gata, Mamsa-gata*, and *Medo-gata Kushta*.[17]

In *Alpa dosha avastha* (mild doshic vitiation), *prachanna karma*—a superficial form of bloodletting performed by gentle scarification with a coarse instrument—is indicated, as there is minimal involvement of both *dosha* and *dushya*.

Conversely, in *Bahu dosha avastha* (marked doshic vitiation), *siravyadha* (venesection) is recommended, as it facilitates the elimination of vitiated blood in conditions where there is extensive involvement of *dosha* and *dushya*.[18]

Shodhana*-based therapeutic protocol in *Kushta*: classical modalities of *Snehana, Vamana, Virechana, Basti, Nasya, Dhumapana,* and *Raktamoksana

I. ***Abhyantara Snehana* (Internal Oleation):** *Mahatiktakaghrita, Mahakhadira ghrita, Panchatikta ghrita, Tiktashatphala ghrita, Triphala ghrita* etc.

II. ***Vamana Karma* (Therapeutic emesis):** *Kutaja, Madanaphala, Madhuka*, along with juice of *Patola* and *Nimba. Shita kashya* (Cold effusion), *Pakva Rasa* (decotion), *Madhu* (Honey) are used for *vaman*a.

III. ***Virechana:*** *Trivruta, Danti*, and *Triphala* should be employed to prepare for *Virechana* in *Kushta. Virechana* in *Kustha* is performed using many forms of *Aushadi Kalpana*, including *Sauviraka* **(**naturally fermented sour gruel**)**, *Tushodaka* (fermented infusion prepared with grain husk), *Alodana* (well-churned liquid preparation), *Asava* (elf-fermented herbal formulation), and *Sidhu* (fermented sugarcane-based alcoholic preparation). Further *Samsarjana Karma* should be performed in accordance with the purification.

IV. ***Asthapana (Niruha) Basti* and *Anuvasana Basti*[19]:** For the administration of *Asthapana (Niruha) Basti*, drugs such as *Darvi, Brihati, Patola, Picchumarda (Nimba tvak), Madanaphala, Kritamala, Kalinga, Yava,* and *Musta* are indicated, processed along with an appropriate quantity of *sneha*. Following *Virechana* and *Asthapana Basti*, if there is persistence of aggravated *Vata, Anuvasana Basti* should be administered. In such conditions, *Sneha Sadita* (Oil or Ghee) prepared by *Madanaphala, Madhuka, Nimba, Kutaja,* and *Patola* is recommended for therapeutic use.

***"Nasthapyah kushtinah"*[20]** *Asthapana Basti* is generally contraindicated in *Kushta*; however, the indication of basti in kushta indicates its utility in Vata-predominant Kushta. Thus, these statements appear to be contradictory in their therapeutic implications.

When features of both indication and contraindication are present simultaneously, the physician should judiciously evaluate their relative severity (guru–laghava) and then arrive at an appropriate therapeutic decision.[21]

V. ***Nasya:*** For *Nasya karma*, drugs such as *Saindhava, Danti, Marica, Phanijihaka, Pippali, Karanjaphala,* and *Vidanga* are indicated, particularly in conditions of *Krimi* and *Kapha*-predominant Kushta**.**

VI. ***Dhumapana[22]:*** *Vairecanika Dhuma* is indicated in *Krimija Kushta*, employing drugs such as *Aparajita, Jyotishmati, Haratala, Manashila, Agaru,* and *Tejapatra* for therapeutic fumigation.

VII. ***Raktamokshana*[23]**

i. In lesions that are *Sthira* (stable) and *Kathina* (indurated), *Swedana* in the form of *prastara sveda* or *naḍi sveda* is indicated, followed by Pracchanna (a form of *raktamokshana*). The patches are gently scarified using a *kurcha* to facilitate localized bloodletting from the affected area.

ii. In elevated lesions, *Swedana* should be performed using a *Sukhoshna pottali* prepared from the meat of *anupa* animals, followed by bloodletting through superficial scarification with a *Tiksna shastra.* In such cases, *Shringa* (horn) or *Alabu* (gourd) may also be employed for *raktamokṣhaṇa*.

Particularly in Alpa kushta, *pracchanna, virecana*, and the application of *Jalauka* (leech therapy) are recommended. The therapeutic efficacy of *lepa* formulations described in *Kushta* is enhanced when applied subsequent to *Raktamokshana* and other *Shodhana* procedures.

VIII. ***Kshara Prayoga in Kusta*[24]:** In conditions characterized by *Sparshanendriya nasha* or *Tvak-indriya hani* (loss of cutaneous sensation), where *Shastra karma* (surgical intervention) is contraindicated, *Kshara karma* is recommended following the elimination of vitiated *Rakta* and *Dosha***.**

IX. ***Agada Prayoga in Kushta*[25]:** In cases of *Kushta* presenting as *Pashana-kathina* (stone-hard), associated with *Suptata* (numbness), *Sthira* (non-spreading), and *Purana* (chronic) lesions, *Agada* (antidotal formulations) should be administered internally prior to the external application of *Visha* in the form of *lepa***,** followed again by the use of *Agada* as a

protective measure.

Shamana Chikitsa

Shamana therapy forms an important component in the management of *Kushta*. After completion of *Sodhana, Shamana Cikitsa* is administered to pacify the residual *Doshas*. It is also useful in patients who are unfit or contraindicated for *Samsodhana.* Acarya Caraka recommends the use of *Tikta* and *Kashaya Dravyas* as *Shamana* drugs following appropriate *Sodhana* and has described several formulations in the *Cikitsasthana.*[26]

Lepa and other external therapies play an important supportive role in the management of *Kitibha Kuṣhta* by reducing *kandu* (itching), *rukshata* (dryness), and *shyavata* (discoloration). *Lepa* prepared with *kushtaghna*, *kandughna*, and *krimighna* drugs having *tikta* and *kashaya rasa* helps in decreasing scaling, inflammation, and pruritus. Drugs such as *Nimba, Haridra, Khadira*, and *Gandhaka* are commonly used in suitable bases according to doshic predominance. Other procedures like *abhyanga* and *parisheka* with medicated oils and decoctions aid in softening the lesions and improving local circulation, thereby enhancing the overall therapeutic outcome.

Shamanoushadis

i. ***Rasoushadi/ Bhasma:*** *Matra: 125-250mg, Anupana: Madhu, Goghrita/ Gomutra, Amalaki swarasa. 1) Shuddha Gandhaka, 2) Rasa Manikya, 3) Gandhaka Rasayana, 4) Talakeshwara rasa, 5) Vydhiharana rasa, 6) Swarna makshika Bhasma, 7) Shuddha Shilajatu, 8) Galatkustari Rasa, 9) Kushta Kutara rasa.*

ii. ***Vati/ Guggulu:*** *Matra: 250-500mg, Anupana: Madhu, Ushnodaka. 1) Arogyavardhini Vati. 2) Yogaraj Guggulu, 3) Shashilekha Vati, 4) Kaishora Guggulu, 5) Panchatikta ghrita guggulu.*

iii. ***Churna:*** *Matra: 3-6 grams, Anupana: Ushna jala, 1) Bakuchi Churna (Somaraji Churna), 2) Manjistadi churna, 3) Pancha nimba Churna, 4) Narasimha Churna, 5) Mustadi Churna, 6) Triphaladi Churna.*

iv. ***Kwath:*** *Matra: 20-40ml, Anupana: Madhu, Gomutra, Ghrita etc 1) Brihat Manjisthadi Kwatha, 2) Patoladi Kwatha, 3) Khadirastaka kwatha.*

v. ***Asava/ Arista:*** *Matra: 20-40ml, Anupana: equal quantity of water, 1) Khadirarishta, 2) Sarivadyasava, 3) Kanakabindvarishta, 4) madhvasava, 5) Triphalasava.*

vi. ***Ghrita:*** *Matra: 10-20ml, Anupana: Kshira, Jala, 1) Mahatiktaka Ghrita, 2)*

Mahakhadira Ghrita, 3) Panchatikta Ghrita, 4) Triphala Ghrita.

vii. ***Taila:*** *1) Bakuchi Taila, 2) Kanakaksheeri Taila, 3) Chaulamoogara Taila, 4) Kushtharakshasa Taila, 5) Somaraji Taila, 6) Marichyadi Taila, 7) Karanja-Nimba Taila.*

viii. ***Paka/ Leha:*** *Matra: 10-20grams, Anupana: Dugdha (milk) Amruta bhallataka leha, 2) Bhallataka avaleha, 3) Dhatryavaleha, 4) Shashankalekhadi Leha.*

ix. **Single Drugs used in *Kushta Chikitsa:*** *1) Manjista, 2) Sariva, 3) Khadira, 4) Haritaki, 5) Vibhitaki, 6) Amalaki, 7) Haridra, 8) Bilva, 9) Vidanga, 10) Aragwadha, 11) Karaveera, 12) Jati patra, 13) bakuchi* etc.

x. **Pralepa:** *1) Manahshiladi lepa, 2) Karanjadi Lepa, 3) Karaveeradi Lepa, 4) Haritakyadi Lepa.*

xi. ***Rasayana Chikitsa:*** *1) Gandhaka Rasayana, 2) Somaraji Rasayana, 3) Haritaki Rasayana, 4) Tuvaraka Rasayana, 5) Bhallataka Rasayana, 6) Haridra Rasayana, 7) Shilajatu Rasayana.*

***Pathya* (Wholesome regimen)**

The recommended wholesome diet and regimen include *Jeerna shali, shastika shali, purana dhanya, yava, godhuma, kodrava, shyamaka, mudga, masura, patola, tikta-rasa shaka, mudga yusha, kulattha yusha, jangala mamsa, laghu anna, ghrita, ushnodaka, bhallataka, triphala, khadira, bakuchi, nimba-yukta anna, mathita* and the adoption of *hitakara* (wholesome lifestyle practices).

***Apathya* (Unwholesome regimen)**

Substances and practices to be avoided include *amla rasa, lavana rasa, katu rasa, dugdha, dadhi, anupa mamsa, tila, masa, guru anna, guda* and *divasvapna* (daytime sleep).

3. MODERN MEDICAL PERSPECTIVE ON PLAQUE PSORIASIS AND CURRENT MANAGEMENT STRATEGIES

3.1 Clinical features of Plaque psoriasis

Plaque psoriasis, the most prevalent clinical variant of psoriasis, is characterised by well-demarcated erythematous plaques covered with silvery scales, predominantly involving the extensor surfaces, scalp, and nails, and typically follows a chronic, stable, yet relapsing course that significantly impacts the patient's physical health, psychological well-being, and social functioning.[27]

The disease shows a bimodal age of onset, with an early-onset form occurring in adolescence or early adulthood, often associated with family history and more severe disease, and a late-onset form appearing between 50 and 60 years, usually with a milder course.

3.2 Assessment

It appears as well-defined, raised erythematous plaques of varied size, often coated with silvery-white scales which when removed may cause pinpoint bleeding [Auspitz sign]. Symptoms of psoriasis include the candle grease sign [when scratched, psoriatic scales fall off, revealing a shining candle-like surface] and Kobner phenomenon which causes skin lesions to appear on healthy skin following a skin injury.

Psoriasis is based on the extent of skin involvement, measured as body surface area (BSA), and the severity of clinical features such as erythema, induration, and scaling. In secondary care settings, validated instruments including the Psoriasis Area and Severity Index (PASI) and the Physician Global Assessment (PGA) are routinely employed, alongside patient-reported outcome measures such as the Dermatology Life Quality Index (DLQI).

1. PASI score.[27]

Plaque Characteristic	Lesion Severity Score	Area involved for each body region affected i.e. Area Score	Amount of body surface area represented by the region
1. Erythema 2. Induration\Thickness 3. Scaling	0 – None 1 – Mild 2 – Moderate 3 – Severe 4 – Very sever	0 – 0% 1 – 1-9% 2 – 10-29% 3 – 30-49% 4 – 50-69% 5 – 70-89% 6 – 90-100%	0.1 – Head and Neck 0.2 – Upper limbs 0.3 – Trunk 0.4 – Lower limbs

PASI Score Calculation

1. Head and Neck (H) – $0.1(E_H + I_H + S_H) A_H$
2. Upper limbs (U) – $0.2(E_U + I_U + S_U) A_U$
3. Trunk (T) – $0.3(E_T + I_T + S_T) A_T$
4. Lower limbs (L) – $0.4(E_L + I_L + S_L) A_L$

E – Erythema

I – Induration/ Thickness

S – Scaling

PASI Score = Sum of H + U + T + L Minimum = 0; Maximum =72

3.3 Treatment/ Management of Plaque Psoriasis[28]

Management of psoriasis encompasses topical therapies such as vitamin D analogues and

corticosteroids; phototherapy modalities including narrowband ultraviolet B (NB-UVB) and psoralen plus ultraviolet A (PUVA); conventional systemic agents like methotrexate, ciclosporin, and acitretin; targeted biologic therapies comprising tumor necrosis factor (TNF), interleukin-17 (IL-17), and interleukin-23 (IL-23) inhibitors; as well as oral small-molecule inhibitors such as dimethyl fumarate and apremilast.[27]

Treatment is individualized according to disease severity, PASI/BSA, lesion site, quality of life, and comorbidities, with goals of lesion clearance, symptom control, relapse prevention, and reduction of systemic risk.

i. **Topical therapy** is first-line for mild disease (BSA <5%).

- Topical corticosteroids (potency based on site and plaque thickness) are most effective but require limited duration to avoid atrophy, telangiectasia, and rebound.
- Vitamin D analogs (calcipotriol /calcitriol) reduce keratinocyte proliferation and are commonly combined with steroids.
- Tazarotene, coal tar, anthralin, and salicylic acid act as antiproliferative and keratolytic agents, improving scaling and plaque thickness.

ii. **Phototherapy** is indicated for moderate disease or inadequate topical response.

- Narrowband UVB is preferred for efficacy and safety.
- Excimer laser is useful for localized plaques.
- PUVA is effective but limited by cumulative phototoxicity and carcinogenic risk.

iii. **Systemic non-biologic therapy** is used for moderate–severe psoriasis (BSA >10%, PASI>10), rapid flares, or special sites (nails, scalp, palms, soles).

- Methotrexate: cornerstone drug with folate supplementation and laboratory monitoring.
- Cyclosporine: rapid control for severe/unstable disease but nephrotoxic with long-term use.
- Acitretin: useful in hyperkeratotic and pustular variants; strongly teratogenic.
- Apremilast and JAK inhibitors: oral targeted therapies with favorable safety and minimal monitoring.

iv. **Biologics** are first-line for moderate–severe disease requiring systemic therapy. They target TNF-α, IL-17, IL-12/23, and IL-23, providing rapid, sustained clearance and improved quality of life.

- IL-17/IL-23 inhibitors → highest skin clearance

- TNF inhibitors and ustekinumab → preferred in psoriatic arthritis

Management follows a stepwise escalation (topical → phototherapy → systemic → biologic) with long-term maintenance and trigger control.

4. PHYTOCHEMICAL AND MOLECULAR BASIS OF SELECTED AYURVEDIC DRUGS IN THE MANAGEMENT OF PLAQUE PSORIASIS

Ayurvedic drugs employed in *Kushta chikitsa* contain diverse phytoconstituents with established immunomodulatory, anti-inflammatory, antiproliferative, and antioxidant activities. This review integrates the phytochemistry of key classical agents—*Haridra* (curcumin), *Bakuchi* (psoralen), *Nimba* (nimbolide), *Guggulu* (guggulsterone), *Khadira* and *Triphala* (catechins and gallic acid), *Manjishtha*, and *Shilajatu* (fulvic acid)—with molecular mechanisms relevant to psoriasis. Experimental and clinical evidence indicates that these compounds modulate central pathogenic pathways, including inhibition of NF-κB, STAT3, and MAPK signalling, suppression of TNF-α, IL-17, and IL-23, regulation of keratinocyte hyperproliferation, and enhancement of cellular antioxidant defenses. Collectively, these multi-target actions provide a mechanistic basis for their traditional therapeutic role and support their potential integration into psoriasis management strategies.

4.1 Phytochemical Classes Relevant to Anti-Psoriatic Activity

- **Polyphenols (Curcumin, Gallic acid)**

Polyphenols exhibit anti-inflammatory and antiproliferative effects through inhibition of NF-κB, STAT3, and pro-inflammatory cytokines in keratinocytes. Curcumin suppresses keratinocyte proliferation and NF-κB/STAT3 signalling[29] Gallic acid reduces oxidative stress and inflammatory mediators.[30]

- **Flavonoids (Quercetin, Catechins)**

Flavonoids down-regulate TNF-α, IL-6, and IL-17 and inhibit MAPK and NF-κB signaling in inflammatory skin models. Quercetin inhibits pro-inflammatory cytokines and oxidative stress[31] Catechins suppress keratinocyte activation and inflammatory pathways.[32]

- **Furanocoumarins (Psoralen)**

Psoralen induces DNA crosslinking under UVA, leading to reduced keratinocyte hyperproliferation and clinical improvement in psoriasis (PUVA mechanism).[33,34]

- **Terpenoids (Nimbolide)**

Terpenoids from *Azadirachta indica* inhibit NF-κB, MAPK, and pro-inflammatory cytokines,

key pathways in psoriasis immunopathology.[35] (Used mechanistically for NF-κB/cytokine suppression relevant to psoriasis inflammation.)

- **Steroidal Compounds (Guggulsterone)**

Guggulsterone inhibits NF-κB activation and down-regulates inflammatory gene expression, including TNF-α and COX-2.[36]

- **Fulvic Acid Complexes (Shilajit)**

Fulvic acid exhibits antioxidant activity, enhances macrophage function, and reduces inflammatory mediators, supporting immunomodulation.[37,38]

4.2 Drug-wise phytochemical and molecular correlation

- ***Haridra* (*Curcuma longa*) – Curcumin**

Curcumin suppresses NF-κB activation, inhibits STAT3 phosphorylation, and reduces TNF-α, IL-17, and IL-23 levels in keratinocytes and immune cells.[39] It also down-regulates COX-2 and iNOS, reducing inflammatory mediators. Clinical studies have demonstrated PASI score reduction with topical and oral curcumin formulations.[40] **Mechanisms:** NF-κB inhibition, STAT3 down-regulation, MAPK pathway modulation, Antioxidant ROS scavenging.

- ***Bakuchi* (*Psoralea corylifolia*) – Psoralen, Bakuchiol**

Psoralen intercalates with DNA and, upon UV exposure, normalizes keratinocyte proliferation, similar to PUVA therapy.[41] Bakuchiol exhibits anti-inflammatory and antiproliferative effects via NF-κB inhibition. **Targets:** Keratinocyte cell cycle arrest, Reduced epidermal hyperplasia, Down-regulation of IL-6 and TNF-α.

- ***Nimba* (*Azadirachta indica*) – Nimbolide**

Nimbolide inhibits NF-κB and MAPK signalling, leading to decreased TNF-α, IL-1β, and IL-6.[42]

It also exhibits antioxidant and anti-proliferative effects on keratinocytes.

- ***Guduchi* (*Tinospora cordifolia*) - tinosporide, cordifolide, magnoflorine**

Guduchi contains diterpenoid lactones (tinosporide, cordifolide)**,** alkaloids (magnoflorine)**,** and immunomodulatory polysaccharides, which suppress nuclear factor kappa B signaling and reduce tumor necrosis factor alpha, interleukin-1 beta, and interleukin-6. It modulates the interleukin-23 and T helper 17 axis and enhances antioxidant enzymes, thereby reducing

reactive oxygen species–mediated keratinocyte activation.[43,44,45]

- ***Guggulu* (*Commiphora mukul*) – Guggulsterone**

Guggulsterone modulates the farnesoid X receptor (FXR) and suppresses NF-κB signalling, reducing inflammatory cytokines.[46] It also improves dyslipidemia, a common psoriasis comorbidity.

- ***Khadira / Triphala* – Catechins, Gallic Acid, Ellagic Acid**

These polyphenols exhibit strong antioxidant activity, inhibit lipid peroxidation, and suppress pro-inflammatory cytokines.[47] Gallic acid reduces keratinocyte proliferation and oxidative stress.

- ***Manjishtha* (*Rubia cordifolia*) – Anthraquinones (Alizarin, Purpurin)**

Demonstrates anti-inflammatory, anti-oxidant, and microcirculatory effects. Experimental studies show inhibition of nitric oxide and pro-inflammatory cytokines.[48]

- ***Shilajatu* – Fulvic Acid**

Fulvic acid modulates macrophage activity, reduces oxidative stress, and improves mitochondrial function, contributing to immunoregulation.[49]

- ***Sariva* (*Hemidesmus indicus*) - Saponins, hemidesmin, flavonoids**

Sariva suppresses nuclear factor kappa B activation and reduces tumor necrosis factor alpha and interleukin-6 production. Its antioxidant flavonoids enhance superoxide dismutase and catalase activity, thereby decreasing reactive oxygen species–mediated keratinocyte activation and modulating immune responses relevant to psoriatic inflammation.[50]

- ***Bilva* (*Aegle marmelos*) - Coumarins (marmelosin), alkaloids, tannins**

Bilva inhibits cyclooxygenase and lipoxygenase pathways, leading to reduced prostaglandin synthesis and decreased pro-inflammatory cytokine release. Its polyphenolic tannins provide antioxidant effects that help normalize inflammatory signaling in epidermal cells.[51]

- ***Vidanga* (*Embelia ribes*) – Embelin**

Embelin inhibits nuclear factor kappa B signalling and reduces expression of tumor necrosis factor alpha and interleukin-1 beta. It also exhibits antioxidant activity and suppresses inflammatory enzyme pathways, contributing to reduced keratinocyte proliferation and inflammatory mediator release.[52]

- ***Aragwadha* (*Cassia fistula*) - Anthraquinones, flavonoids**

Aragwadha reduces oxidative stress and down-regulates pro-inflammatory cytokines through inhibition of nuclear factor kappa B–mediated transcription. Its flavonoids contribute to immunomodulation and decreased inflammatory cell activation in psoriatic pathways.[53]

- ***Karaveera* (*Nerium indicum*)- Cardiac glycosides (oleandrin)**

Oleandrin demonstrates antiproliferative effects on keratinocytes through modulation of cellular signalling pathways involved in cell cycle regulation and inhibition of inflammatory mediator production. It also interferes with nuclear factor kappa B–dependent transcription, though its use requires strict dose regulation due to potential toxicity.[54]

- ***Jati Patra* (*Jasminum officinale*) - Flavonoids, secoiridoids, essential oils**

Jati Patra reduces nitric oxide production and suppresses pro-inflammatory cytokines by inhibiting inducible nitric oxide synthase and nuclear factor kappa B signalling. Its antioxidant constituents reduce reactive oxygen species and support normalization of inflammatory responses in skin tissue.[55]

- **Gandhaka Rasayana**

Gandhaka Rasayana, composed of purified elemental sulfur (S_8) processed through ***s**hodhana* and Bhavana, generates hydrogen sulfide (H_2S) and reactive sulfur species in vivo. In plaque psoriasis, it acts through keratolytic effects (disrupting keratin disulfide bonds to reduce scaling), NF-κB inhibition with suppression of TNF-α and IL-17 pathways (controlling Th17-mediated inflammation), and glutathione-mediated antioxidant activity (reducing oxidative stress). Thus, it targets keratinocyte hyperproliferation, immune dysregulation, and inflammation, correlating with its Ayurvedic *Kushtaghna* and *Raktaprasadana* properties.[56,57,58,59]

5. DISCUSSION

The present review critically correlates the Ayurvedic concept of *Kitibha Kushta* with the contemporary understanding of plaque psoriasis, highlighting strong conceptual and mechanistic parallels. Classical descriptions of *Shyava varna, Kinakhara/Khara sparsha, Parusha, and Ugra kandu* closely resemble the erythematous, scaly, indurated plaques and pruritus characteristic of psoriasis. The Ayurvedic explanation of *Tridosha dushti*—predominantly *Vata–Kapha* with *Rakta involvement*—finds biomedical correlation in

keratinocyte hyperproliferation, immune dysregulation (Th17 axis activation), dermal vascular changes, and chronic inflammatory cytokine cascades (TNF-α, IL-17, IL-23).

The described *Samprapti* involving *Agnimandya*, *Ama formation*, *Rasarakta vaha srotodushti*, and *Dosha–Dushya sammurchana* parallels modern concepts of metabolic inflammation, oxidative stress, gut–skin axis disturbance, endothelial activation, and immune–keratinocyte feedback amplification. The chronic, relapsing (*Chirakari*) nature of *Kitibha Kushta* further aligns with the relapsing–remitting course of psoriasis.

Therapeutically, Ayurveda emphasizes a multimodal strategy: *Nidana parivarjana*, *Shodhana*, *Shamana*, external applications, and *Rasayana*. *Shodhana* procedures such as *Virechana* and *Raktamokshana* are conceptually comparable to systemic detoxification and inflammatory load reduction, while Shamana formulations target immune modulation and tissue stabilization. Importantly, the phytochemical and molecular review of selected drugs—including curcumin, psoralen, nimbolide, guggulsterone, catechins, fulvic acid, and Sulphur derivatives—demonstrates inhibition of key inflammatory pathways such as NF-κB, STAT3, MAPK, and suppression of TNF-α and IL-17. These mechanisms directly address the central immunopathogenesis of psoriasis.

Gandhaka Rasayana merits special attention due to its Sulphur-mediated keratolytic action, NF-κB inhibition, Th17 pathway modulation, and glutathione-enhancing antioxidant effect, thereby targeting hyperproliferation, inflammation, and oxidative stress simultaneously. The integration of PASI scoring provides an objective, standardized clinical assessment tool to evaluate Ayurvedic interventions in a measurable and globally accepted format.

6. CONCLUSION

The present article comprehensively reviews *Kitibha Kushta* with special reference to plaque psoriasis. *Kitibha Kushta*, elaborated in both *Brihattrayi* and *Laghutrayi* under the *Kuṣṭha Prakarana*, is clinically relevant even today. In contemporary practice, *Kitibha Kushta* can be correlated with several chronic dermatoses, such as psoriasis, dry eczema, lichen simplex chronicus, and prurigo nodularis. Among these, the morphological features of *Kitibha Kushta* show a close resemblance to plaque psoriasis. Furthermore, analysis of *Dosha–Dhatu–Mala–Srotas* involvement supports this correlation. Therefore, understanding plaque psoriasis through the Ayurvedic perspective of *Kitibha Kushta* facilitates a rational and comprehensive management approach.

Plaque psoriasis, being an immune-mediated chronic inflammatory condition, poses limitations for complete cure. Long-term use of corticosteroids and immunosuppressive agents is often associated with adverse effects, negatively impacting patients' quality of life and predisposing them to future complications. Ayurvedic management aims not only at symptom control but also at reducing relapses and maintaining remission through comprehensive therapeutic approaches, including *Rasayana* therapy.

Overall, the evidence suggests that Ayurvedic therapeutics exert multi-target, immunomodulatory, antiproliferative, and antioxidant actions, which are mechanistically coherent with contemporary psoriasis biology. This integrative framework strengthens the scientific validity of classical principles and supports further systematic clinical research.

ACKNOWLEDGEMENT

The authors sincerely acknowledge the invaluable contributions of classical Ayurvedic scholars whose foundational texts provide the basis of this work, and modern researchers whose scientific studies and publications have enriched the contemporary understanding of the subject.

AUTHOR CONTRIBUTIONS

Conceptualization, literature review, data analysis, manuscript drafting, critical revision for important intellectual content, and final approval of the version to be published.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this paper.

7. REFERENCES

1. Vaghbhata. *Ashtanga Hridaya* with Sarvangasundara commentary of Arunadatta and Ayurvedarasayana of Hemadri. Ed. Bhishagacharya Harishastri Paradakara Vaidya. Reprint ed. Varanasi: Chaukhambha Orientalia; 2025. Nidana Sthana, Ch. 14, Ver. 4, 524.
2. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia; 2023. Chikitsa Sthana, Chapter. 7, Verse. 22, 451.
3. Sushruta. *Sushruta Samhita* with Nibandha Sangraha commentary of Dalhana and Nyayachandrika Panjika of Gayadasa on *Nidana Sthana*. Edited by Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Choukhambha Orientalia; 2023. Nidana Sthana, Chapter.

5, Verse. 14., 286.

4. Vaghbhata. *Ashtanga Hridaya* with Sarvangasundara commentary of Arunadatta and Ayurvedarasayana of Hemadri. Ed. Bhishagacharya Harishastri Paradakara Vaidya. Reprint ed. Varanasi: Chaukhambha Orientalia; 2025. Nidana Sthana, Ch. 14, Ver. 20., 525.
5. Madhavakara. *Madhava Nidana* (*Madhavanidanam*) with Madhukosa commentary of Vijayarakṣita and Kaṇṭhadatta, and Atankadarpana of Vacaspati Veda. Ed. Trivikrama Atmaja Yadavasarma. Varanasi: Chaukhambha Orientalia, 2002; Ch. 49, Ver. 18., 166.
6. Sushruta. *Sushruta Samhita* with Nibandha Sangraha commentary of Dalhanacharya and Nyayachandrika of Gayadasa. Ed. Yadavaji Trikamji Acharya. Varanasi: Chaukhambha Orientalia, 1997; Nidana Sthana, Ch. 5, Ver. 32–33., 289.
7. Sushruta. *Sushruta Samhita* with Nibandha Sangraha commentary of Dalhanacharya and Nyayachandrika of Gayadasa. Ed. Yadavaji Trikamji Acharya. Varanasi: Chaukhambha Orientalia, 1997; Nidana Sthana, Ch. 5, Ver. 27., 288.
8. Sushruta. *Sushruta Samhita* with Nibandha Sangraha commentary of Dalhanacharya and Nyayachandrika of Gayadasa. Ed. Yadavaji Trikamji Acharya. Varanasi: Chaukhambha Orientalia, 1997; Nidana Sthana, Ch. 5, Ver. 6., 283.
9. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Viman Sthana, Chapter. 7, Verse. 11., 258.
10. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 14, Verse. 179., 508.
11. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 4, Verse. 27., 430.
12. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 19, Verse. 16, 550.
13. Mehta S, Sathe NC. Plaque Psoriasis. [Updated 2025 Sep 14]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing, 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430879/
14. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi:

Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 7, Verse. 41, 452.

15. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 7, Verse. 31, 451.
16. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Sutra Sthana, Chapter. 25, Verse. 40, 132.
17. Sushruta. *Sushruta Samhita* with Nibandha Sangraha commentary of Dalhana and Nyayachandrika Panjika of Gayadasa on *Nidana Sthana*. Edited by Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Choukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 9th, Verse. 6th, 442.
18. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 7, Verse. 40,41, 452.
19. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 7, Verse. 46,47, 452.
20. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Siddhi Sthana, Chapter. 2, Verse. 14, 677.
21. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Vimana Sthana, Chapter. 8, Verse. 134, 272.
22. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Sutra Sthana, Chapter. 5th, Verse.26, 39.
23. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 7, Verse.50-53, 452.
24. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 7, Verse.54. 453.
25. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi:

Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 7, Verse.55. 453.

26. Agnivesha. *Charaka Samhita*, redacted by Charaka and Drudhabala with Ayurveda Dipika commentary. Ed. Yadavaji Trikamji Acharya. Reprint ed. Varanasi: Chaukhambha Orientalia, 2023; Chikitsa Sthana, Chapter. 7, Verse. 58. 453.
27. Ralston SH, Penman ID, Strachan MWJ, Hobson RP, editors. *Davidson's principles and practice of medicine*. 23rd ed. Edinburgh: Elsevier, 2018; Chapter 29, Psoriasis and other erythematous scaly eruptions, 1247–1251.
28. Mehta S, Sathe NC. Plaque Psoriasis. [Updated 2025 Sep 14]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing, Jan. 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430879/
29. Kang S, et al. Curcumin inhibits keratinocyte proliferation via NF-κB and STAT3 pathways. *J Dermatol Sci.*, 2016; 82(2): 85-92.
30. Kahkeshani N, et al. Antioxidant and anti-inflammatory effects of gallic acid. *Nutrients*, 2019; 11(5): 1063.
31. Li Y, Yao J, Han C, et al. Quercetin, inflammation and immunity. *Nutrients*, 2016; 8(3): 167.
32. Katiyar SK. Green tea catechins and skin photoprotection and inflammation. *Arch Biochem Biophys*, 2011; 508(2): 152-158.
33. Parrish JA, Fitzpatrick TB, Tanenbaum L, Pathak MA. Photochemotherapy of psoriasis with oral methoxsalen and UVA. *N Engl J Med.*, 1974; 291(23): 1207-1211.
34. Stern RS. PUVA follow-up study: mechanism and efficacy in psoriasis. *J Am Acad Dermatol*, 2001; 44(5): 755-761.
35. Gupta SC, et al. Nimbolide suppresses NF-κB and inflammatory signaling pathways. *Mol Cancer Ther.*, 2010; 9(6): 1636-1645.
36. Shishodia S, Aggarwal BB. Guggulsterone inhibits NF-κB activation and inflammatory gene products. *J Biol Chem.*, 2004; 279(45): 47148-47158.
37. Ghosal S. Shilajit in perspective. *Phytother Res.*, 1995; 9(5): 328-332.
38. Carrasco-Gallardo C, Guzmán L, Maccioni RB. Shilajit: a natural phytocomplex with potential anti-inflammatory and antioxidant effects. *Int J Alzheimers Dis.*, 2012; 2012: 674142.
39. Kang S, et al. Curcumin inhibits keratinocyte proliferation and NF-κB signaling. J Dermatol Sci., 2016; 82: 85-92.
40. Kurd SK, et al. Oral curcumin in psoriasis: A randomized trial. J Am Acad Dermatol, 2008; 58: 625-631.

41. Parrish JA, et al. Photochemotherapy of psoriasis with psoralens. N Engl J Med., 1974; 291: 1207-1211.
42. Gupta SC, et al. Nimbolide suppresses NF-κB and inflammatory pathways. Mol Cancer Ther., 2010; 9: 1636-1645.
43. Singh N, et al. Immunomodulatory activity of *Tinospora cordifolia*. *J Ethnopharmacol,* 2003; 84(2-3): 199-206.
44. Saha S, et al. *Tinospora cordifolia* inhibits NF-κB activation and inflammatory mediators. *Int Immunopharmacol,* 2012; 14(4): 637-644.
45. Sharma U, et al. Antioxidant and immunomodulatory properties of *Tinospora cordifolia*. *J Ethnopharmacol,* 2012; 141(3): 918-926.
46. Shishodia S, et al. Guggulsterone inhibits NF-κB activation. J Biol Chem., 2004; 279: 47148-47158.
47. Sandhir R, et al. Antioxidant activity of Triphala. Phytother Res., 2010; 24: 1347-1352.
48. Deoda RS, et al. Anti-inflammatory activity of *Rubia cordifolia*. J Ethnopharmacol, 2011; 134: 531-537.
49. Ghosal S, et al. Fulvic acid in Shilajit: antioxidant and immunomodulatory effects. Phytother Res., 1995; 9: 328-332.
50. Das S, et al. Anti-inflammatory activity of *Hemidesmus indicus*. *J Ethnopharmacol,* 2011; 136: 283-287.
51. Sharma GN, et al. Anti-inflammatory and antioxidant activity of *Aegle marmelos*. *J Ethnopharmacol,* 2011; 133: 111-115.
52. Chitra M, et al. Embelin inhibits NF-κB and inflammatory pathways. *Biochem Pharmacol,* 1994; 48: 1177-1184.
53. Yadav R, et al. Anti-inflammatory activity of *Cassia fistula*. *J Ethnopharmacol,* 2010; 128: 362-368.
54. Newman RA, et al. Oleandrin-mediated antiproliferative activity. *J Exp Ther Oncol,* 2008; 7: 333-342.
55. Ali B, et al. Anti-inflammatory and antioxidant activity of *Jasminum officinale*. *J Ethnopharmacol,* 2014; 151: 298-306.
56. Wallace JL, Wang R. Hydrogen sulfide-based therapeutics: exploiting a unique but ubiquitous gasotransmitter. *Nat Rev Drug Discov,* 2015; 14(5): 329-345.
57. Lowes MA, Bowcock AM, Krueger JG. Pathogenesis and therapy of psoriasis. *Nature.,* 2007; 445: 866-873.
58. Briganti S, Picardo M. Antioxidant activity, lipid peroxidation and skin diseases. *J Eur*

Acad Dermatol Venereol, 2003; 17(6): 663-669.

59. Mohan R et al. Pharmaceutical standardization of Shuddha Gandhaka. *AYU.*, 2014; 35(3): 311-315.